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Aortic valve replacement with frame-mounted autologous fascia lata
Long-term results[1]

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Between June 1969 and August 1970, 80 patients, aged 22 to 63 years, had lone aortic valve replacement with frame-mounted autologous fascia lata. The operative mortality was 3·8 per cent. Seventy-seven survivors have been followed for 3 to 4 years and 50 have had postoperative investigation. There have been 8 subsequent deaths, 3 from infective endocarditis, 4 from aortic regurgitation, and one unknown. Fifteen patients developed late aortic regurgitation. Seven required reoperation and valve tissue was found to have torn from the supporting frame in 6 patients. Serial investigations and clinical examination showed no evidence of progressive obstruction occurring in the valves. Late systemic emboli occurred in 9 patients. In view of the development of serious aortic regurgitation in survivors after 2 years and the occurrence of systemic emboli, this form of aortic valve replacement cannot be considered to be satisfactory in the long term, in comparison with other forms of biological or mechanical valve replacement.

The use of autologous fascia lata for replacement of the aortic valve has been established (Senning, 1967, 1968; Edwards, 1969; Edwards *et al.*, 1969; Ionescu and Ross, 1969). Early reports have suggested that this provided a satisfactory aortic valve substitute. The benefits of using biological material for valve replacement have been accepted (Ross, 1967) and it was hoped that the use of autologous fascia lata would provide a viable valve (Ionescu and Ross, 1969) and solve the problems of availability of valve tissue.

Senning's technique fashioned unsupported fascial aortic valves and provided in his experience reasonable functional results, but in this series frame-mounted fascia has been used. Now that postoperative problems in relation to cardiopulmonary bypass for valve replacement have been solved, the true value of a new form of valve substitute can only be assessed by long-term results.

We report here the results in 77 survivors 3 to 4 years after lone aortic valve replacement by a frame-mounted valve made of autologous fascia lata removed from the thigh.

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Subjects and methods.

Between June 1969 and August 1970, 80 patients had isolated aortic valve replacement with frame-mounted autologous fascia lata at the National Heart Hospital, London. These represent all patients seen at the hospital during this period who required lone aortic valve replacement, except for a few emergency operations where prosthetic valves were used to enable the operation to be made as quickly as possible and 8 where unsupported fascial valves were used. However, the latter were found to be unsatisfactory and were soon abandoned in favour of supported fascial valves. Patients were aged 22 to 63 with an average age of 47·2 years. Sixty-four were male and 16 were female. Twenty-seven had congenital aortic valve disease, and 42 had rheumatic disease; there were single examples of Marfan's syndrome and syphilitic aortic valve disease and in 9 patients the aetiology was uncertain, but probably congenital. Thirty-five patients had dominant aortic stenosis, 32 had dominant regurgitation, and 9 had a mixed lesion. In 4 patients the indication for operation was haemolysis from a regurgitant Starr-Edwards prosthesis. Twelve patients had had previous aortic valve replacement and one an open aortic valvotomy. Ten patients had additional mitral valve disease, one of whom also had tricuspid disease.

Thirty-six patients had been in recent left ventricular

failure and one had active infective endocarditis with low cardiac output state. All had important left ventricular hypertrophy. Three patients had had recently treated infective endocarditis. Four patients had additional clinical coronary artery disease.

Methods

After a strip of fascia lata was removed from the lateral aspect of the thigh, the valve was prepared on a frame as previously described (Ionescu and Ross, 1969; Ionescu *et al.*, 1970a, b). Normothermic cardiopulmonary bypass was used in all patients and the length of bypass varied from 48 to 139 minutes; intermittent coronary perfusion was used and the total period of ischaemic arrest was between 7 and 80 minutes. The diameter of the frame was 20 mm in 22 patients, 22 mm in 53, and 24 mm in 5.

Seventeen patients required additional procedures; 4 an open mitral valvotomy, 2 mitral valve repair, and one had a mitral replacement with Starr prosthesis and tricuspid annuloplasty, 3 patients had a subvalvular myotomy, and a further 2 had obliteration of unruptured aneurysms of a sinus of Valsalva. Three had closure of ventricular septal defects which were caused by infection on the aortic valve in 2. One patient had an ascending aortic 'dacron' graft replacement and another with active syphilis required a coronary patch for coronary ostial stenosis.

Patients were clinically assessed after operation, before leaving hospital, and were seen in the outpatient department 3 and 6 months later and subsequently at 6-monthly intervals. Twenty-two patients have been followed at other centres and we have relied on data from referring physicians. Anticoagulants were only given for the first 3 months and then stopped; the thrombotest was used to control the anticoagulation and the level was maintained between 5 and 15 per cent.

Assessment was made of symptoms, regression of left ventricular hypertrophy, and the development of signs of aortic stenosis and regurgitation. Particular attention was paid to the appearance of new murmurs and an immediate diastolic murmur was accepted as evidence of aortic regurgitation. In addition to chest radiography and electrocardiograms, screening was carried out intermittently for valve calcification. Between August 1969 and March 1970 cardiac catheterization was undertaken on 14 patients within 8 weeks of operation. Late catheterization was performed in 43 patients between 4 and 39 months after operation. Seven patients were catheterized both early and late, and 4 patients were catheterized twice in the 'late' period, giving a total of 11 patients who were studied on 2 occasions.

Results

Hospital mortality

Three patients died in the first 2 weeks, an operative mortality of 3·8 per cent. One with active syphilis did not recover consciousness after operation and 2 died from renal failure.

Late mortality

There have been 8 late deaths over the 3- to 4-year period giving a late mortality of 10·4 per cent (Table 1). The first was a patient operated on during active infection who died 2 months later after a second operation for persistence of aortic regurgitation and a ventricular septal defect. Aortic regurgitation was peripheral due to a dehiscence of sutures, and the cusps looked healthy. Three patients died from the effects of active infective endocarditis, 2 at 5 months and 1 at 34 months. One patient with mild aortic regurgitation died at 7 months after additional mitral valve surgery and another with mild aortic regurgitation died after operation for secondary subvalve obstruction. The seventh patient died at operation for aortic regurgitation at 40 months, and an eighth patient died suddenly 3½ years later having had a good clinical result and mild persistent aortic regurgitation when assessed at 3 years. Necropsy was not performed.

Clinical signs

After valve replacement, it was usual for an aortic ejection sound to be recorded at the apex and aortic and pulmonary closure (A_2 and P_2) were clear. Short ejection systolic murmurs were usually present. Electrocardiographic evidence of left ventricular hypertrophy regressed in 30 per cent (Fig. 1) and the heart size diminished in those where

TABLE 1 *Deaths after aortic valve replacement with autologous fascia lata*

	Case no.	*Time after operation*	*Cause*
Early deaths	4	1 dy	Active syphilis; cerebral damage
	42	9 dy	Acute renal failure
	45	12 dy	Acute renal failure
Late deaths	73	2 mth	Second operation for recurrent aortic regurg. and ventricular septal defect
		5 mth	*Aspergillus fumigatus* endocarditis
	36	5 mth	*Staph. albus* endocarditis
	14	7 mth	Second operation for recurrent aortic regurg. and mitral valve replacement
	65	14 mth	Second operation for recurrent aortic regurg. and subaortic stenosis
	7	34 mth	*Strep. viridans* endocarditis
	40	40 mth	Second operation for aortic regurgitation
	30	42 mth	? Cause

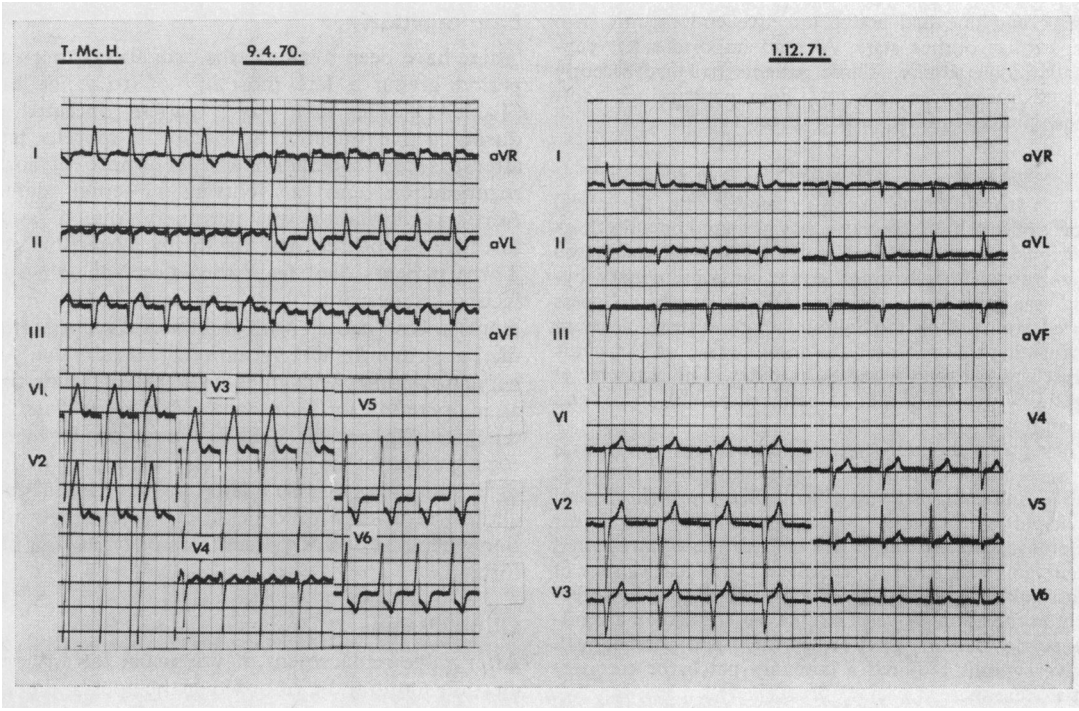

FIG. 1 *Electrocardiogram before (9.4.70) and 20 months after aortic valve replacement with supported fascial valve.*

it had been enlarged, provided aortic regurgitation was not present. In the 6 patients who developed late serious aortic regurgitation, the heart enlarged rapidly having been previously normal for 1 to 2 years after the first operation.

Investigation

Fifty patients had haemodynamic investigation after aortic valve replacement. Cardiac catheterization included measurement of right heart pressures, aortic valve gradient when the valve could be crossed with a catheter, and cineaortography for the assessment of aortic regurgitation.

Fourteen patients were studied in the first 8 weeks, of whom 7 were restudied between 4 and 39 months after operation. The remaining 36 patients were studied during this late period making a total of 43 with late postoperative data. The group which were investigated within 8 weeks included 4 of the 5 patients who developed early severe aortic regurgitation and who underwent reoperation. Also included were 6 patients with mild aortic regurgitation which has not been progressive and was thought to be peripheral.

Of the 15 patients who developed aortic regurgitation after the first year, 10 have been investigated, and serious aortic regurgitation was confirmed in 7, requiring reoperation. In 4 of these, early studies showed that the valve was competent. The fascia remained pliable in the early cases and in those with good function 2 to 4 years later. The cusps appeared to open fully and close normally with the appearance of a normal aortic valve.

In the 50 patients who have been catheterized after operation, systolic valve gradients have been obtained in 37; in 25 of these patients the gradient was less than 10 mmHg and only 4 had a gradient greater than 20 (Table 4); 3 of these 4 patients had a frame size of 20 mm diameter and the fourth had a frame size of 22 mm. In 4 patients only was the gradient measured on 2 occasions and in none of these was there evidence of progressive stenosis.

Aortic regurgitation

Five of the 77 surviving patients had important regurgitation within the first weeks after operation and all required further operation; 3 died after the second operation. In each case regurgitation was peripheral, between the aortic wall and the valve frame and was caused by a dehiscence of sutures.

Six patients left hospital with signs of trivial or moderate regurgitation which has in these cases remained unchanged throughout the follow-up period. Thus, 11 (14% of survivors) developed early regurgitation and 66 patients (86%) left hospital with a clinically competent aortic valve.

Of the 66 patients who left hospital with a clinically competent aortic valve, 15 had developed moderate or severe aortic regurgitation 1 to 4 years later. This does not include 3 patients who developed regurgitation after infective endocarditis. Five developed it in the second year, 9 in the third, and 1 in the fourth year, though so far only 10 patients have been followed for a full 4 years. Of these 15, 10 have been investigated with aortography and 7 have been severe enough to require further valve surgery and 1 died (Table 2). In 6 patients, cusp tissue had torn from the frame at the site of the prong forming the commissure (Fig. 2 and 3). The seventh patient had a shrunken and necrotic left coronary cusp. There are 51 patients (68%) out of the original 74 survivors who have clinically competent valves at follow-up time of 3 to 4 years.

Infection

Postoperative endocarditis occurred in 3 of the 77 survivors (4%), one due to *Aspergillus fumigatus* (*British Medical Journal*, 1970) and another to *Staphylococcus albus*, causing death at 5 to 6 months. It is possible that both these infections might have dated from the time of operation. Both these patients were treated at other centres and were not offered emergency surgery. The third patient developed *Streptococcus viridans* endocarditis at 34 months, which was related to inadequate antibiotic cover during dentistry; he died despite emergency valve replacement; the valve was almost completely destroyed (see pathology of valves).

Emboli

Nine patients developed visual or neurological symptoms suggestive of arterial emboli; 6 patients had transient blurring of vision or diplopia suggesting microemboli to the retinae or brain-stem (Table 3). Three patients have developed hemiplegias which have resulted in permanent damage in 2 patients. One of these 9 patients has suffered from both diplopia and left facial paraesthesia in relation to cardiac catheterization. These complications have not followed any time pattern but have occurred in different patients at any time in the follow-up period. Five patients were not taking anticoagulants at the time and 4 were taking them and appeared to be controlled.

Other complications

After operation 7 patients had electrocardiographic evidence suggesting myocardial infarction. The ischaemic arrest time during operation for these 7 patients varied from 16 to 50 minutes (average 29 minutes) and was similar to the group without infarction. Two patients developed complete heart

TABLE 2 *Patients investigated for late onset of aortic regurgitation*

Case no.	*Onset of aortic regurgitation (mth postop.)*	*Date of aortogram (mth postop.)*	*Severity of aortic regurgitation (0–4)*	*Sequel*	*Pathology*
17	30	30	1	—	—
19	24	24	1–2	Valve replaced at 47 mth	Torn commissure
22	12	32	2	—	—
32	27	27	3	Valve replaced	Torn commissure
34	24	26	2	Valve replaced	Torn commissure
38	19	24	2	—	—
39	24	39	3	Valve replaced	Torn commissure
40	19	24	2	Valve replaced at 40 mth; died	Torn commissure
44	16	20	2–3	Valve replaced at 35 mth	Shrunken cusps
71	39	39	3	Valve replaced	Torn commissure

Note: In addition to the above 10 patients, 5 patients with late onset of aortic regurgitation of clinically mild degree have not been investigated.

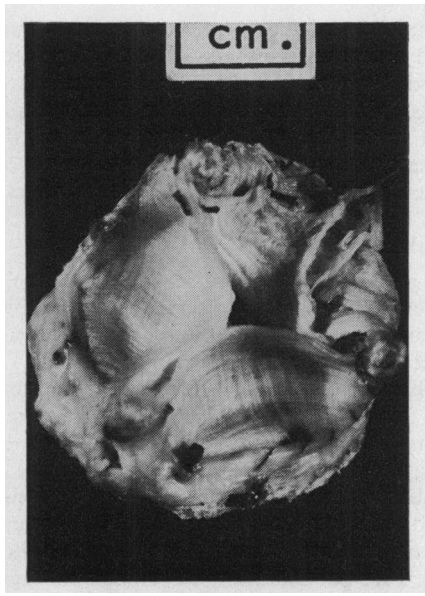

FIG. 2 *Aortic valve removed 28 months after operation. Viewed from aortic surface. Two cusps are normal and one has torn completely from supporting strut.*

block; one of these required permanent pacing, and the other reverted to second-degree block. There were 4 patients who had left bundle-branch block after operation. None of these had a myotomy. One patient had sloughing of the fascial donor site which led to necrosis of the vastus lateralis and some permanent leg disability.

Pathology of valves

Pathological data have been difficult to obtain. In the 3 patients who died in the first 2 weeks, the valves were said to look normal and no histology is available. Histology on a valve removed at 34 months for active endocarditis showed a haemorrhagic abscess with recent fibrinoid necrosis, newly formed blood vessels, and frayed ends of sutures in the obviously infected cusp. In the cusp which was macroscopically free of infection, the tissue was acellular, and polarized light showed that it was built of wavy collagen running in all directions. On one surface there was a fibrous web with bacteria in it and much new fibroblastic tissue has grown in the peripheral edge of the cusps over the frame.

In a valve considered to be clinically competent, examined at 7 months after the patient died from myocardial failure and mitral regurgitation, the cusps were totally acellular, composed of wavy collagen in regular transverse directions, and there was a single layer of endothelium covering it on the left ventricular surface. On the aortic surface, there was a coating of fibrin mesh with mononuclear lymphocytes in it. Also on this surface were crystals of refringent structures with foreign body giant cells.

These ovoid crystal like bodies have been seen in other fascial valves put in at other sites (I. Aarons, 1973, personal communication). The possibility of talc from the surgeon's gloves has been raised. In 3 patients whose valves have torn acutely at the commissural attachment between 2½ and 3 years later, the collagen close to the strut has broken but there is no special change in it and the rest of the valve looks as described above.

Discussion

The operative mortality is low (3·8%) and parallels that for other forms of aortic valve replacement at the National Heart Hospital. However, in assessing any new form of biological valve, concern should be with the long-term function. Though 62 per cent of patients with supported aortic fascial valves have a competent valve 3 to 4 years later, the late mortality of 10 per cent, the late onset of serious aortic regurgitation in 12 per cent 2 to 4 years after operation, and the incidence of late small emboli cause grave concern.

In analysing aortic regurgitation after valve replacement, it is important to distinguish that related to technical problems occurring early, and regurgitation developing from valve failure due to a degenerative change. Eleven patients had early aortic regurgitation, severe in 5 and requiring reoperation; it was found to be caused by suture dehiscence causing peripheral leaking between the frame and the aortic wall, which is related to surgical technique and not to durability of tissue. In the other 6 patients, the source is not established, but cineaortography suggests that it is also peripheral. In the 15 developing late aortic regurgitation after 18 months, it was rapidly progressive in 7 requiring reoperation, and in 6 of them the appearance was the same, namely tearing of the cusp from the commissural strut. These valves were previously competent.

It appears that the supported aortic fascial valves may fail in two ways, either by shrinking and thickening, causing progressive central regurgitation (which has only occurred once), or by sudden rupture. This is not surprising in view of the pathological findings in several valves, for the cusps are totally acellular and composed of collagen fibres in parallel wavy disposition and ruptured at the point of most stress. The progressive shrinkage in one valve seen at necropsy is difficult to understand but is similar

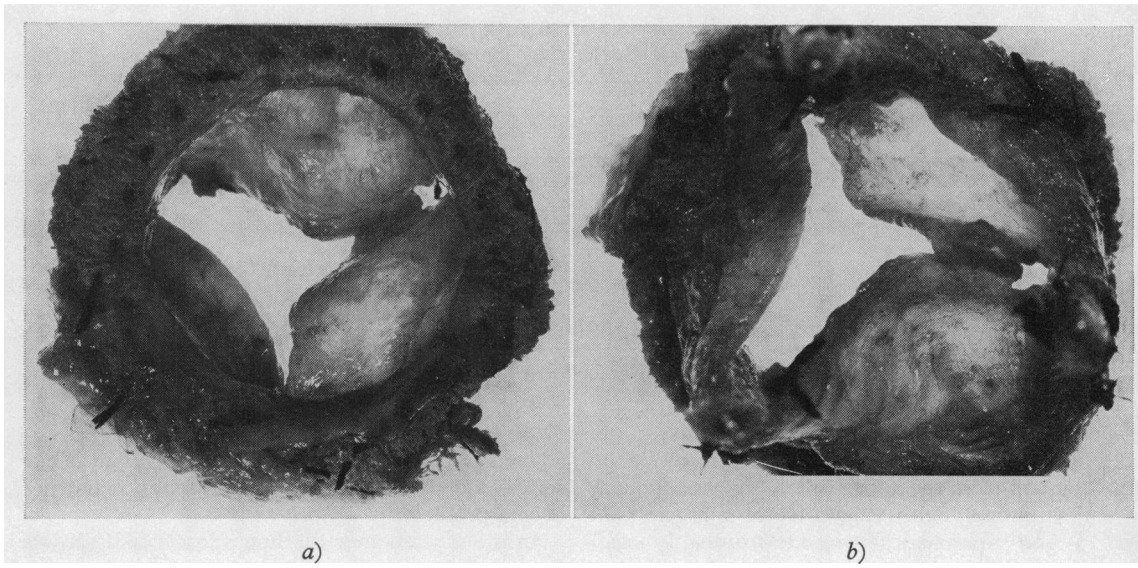

FIG. 3 *a) Ventricular surface of frame-mounted aortic fascial valve removed at surgery after the first operation. There is thickening of one cusp (to the left) and the site of tearing from the strut is shown by the arrow. b) Same valve viewed from the aortic side.*

to that seen in mitral fascial valves (Petch *et al.*, 1974). In one specimen non-circular crystal-like structures were seen where the cusp edge curled, and these were related to a discrete giant cell reaction. These have been seen in other removed fascial valves, and the granules may represent talcum powder from the gloves of the valve maker. Such a foreign body may cause fibrotic reaction. Viability studies on fascia from aortic and mitral valves have shown no evidence of live cells (N. Al-Janabi and R. Parker, 1973, personal communication).

TABLE 3 *Embolic complications after aortic valve replacement with autologous fascia lata in 9 patients*

Case no.	*Months after operation*	*Clinical features*	*Anti-coagulants*
6	18	Transient bilateral blurring of central vision	−
	21	Transient blindness left eye	−
31	21	Transient diplopia	−
	27	Transient diplopia and left facial paraesthesia after cardiac catheterization	−
60	6–12	Several transient episodes of blurred vision	−
57	1–14	Many episodes of diplopia; one transient episode of inferior hemianopia	+
66	23	Four transient episodes of inferior hemianopia	−
80	30	Episodes of diplopia	−
16	16	Transient right hemiplegia	−
	19	Second more severe episode	+*
	28	Right hemiplegia and *grand mal* convulsion	+
21	13	Left hemiplegia	−
68	3	Several transient episodes of left hemiplegia	+

*Anticoagulants started after first embolic episode.

TABLE 4 *Pressure gradients across aortic fascia lata valves in patients with 2 measurements or with gradients greater than 20 mmHg*

Case no.	*Gradient (mmHg)*	*Time after operation*	*Gradient (mmHg)*	*Time (months) after operation*
55	5	3 weeks	5	16
20	10	2 ,,	15	27
19	25	1 months	20	24
5	30	5 ,,	Valve not crossed	35
2	40	9 ,,	5*	35
12	50	5 ,,	—	—

*The decreased gradient in this patient is unexplained.

A further problem is the occurrence of probable small emboli from the aortic valve. Six patients had visual symptoms highly suggestive of microemboli, and 3 had neurological episodes probably also embolic (Table 3). These did not appear to be influenced by anticoagulants; 3 patients were taking anticoagulants at the time of the episode. Their nature suggested microemboli but no platelet aggregates were seen on the few valves histologically examined; however, fibrin webs have been seen on the aortic valve surface in another patient and it is possible that these are minute fibrin emboli.

In spite of these problems, there are good features. Progressive stenosis of these fascia lata valves has not yet occurred and no valve calcification has been noted radiographically. In those where gradients were found, it was related to the small size of the frame (20 mm) and none showed progressive obstruction (Table 4). Another favourable feature is that 62 per cent of survivors were competent 3 to 4 years later. Fascia in the aortic position has functioned better than in the tricuspid or right ventricular outflow where it has been a disaster (Dalichau, Gonzalez-Lavin, and Ross, 1972; Ross and Somerville, 1971) and in the mitral position there is progressive dysfunction after 18 months in many patients (Petch *et al.*, 1974). Dalichau and his colleagues speculated that this difference was related to free movement of all 3 leaflets in the aortic position with high opening and closing pressures. Increasing numbers of ruptured valves are expected in the future in view of the constancy of the macroscopical appearance in those who deteriorate after 2 years.

It is of interest that the incidence of infection has been low (4%); in 2 of the patients it was probably operative making the acquired incidence only 1 to 2 per cent. This low incidence of endocarditis contrasts with the higher incidence reported by Senning (1967). Infection has been considered a major disadvantage of this type of valve in the past, but in our series, and that of Ionescu *et al.* (1972), it has not been a problem.

In the 2 years after the first operation fascia lata appeared to provide a satisfactory valve substitute: hence the initial hopeful reports (Balcon *et al.*, 1970; Gonzalez-Lavin and Ross, 1971; Ionescu *et al.*, 1972). However, a closer look after a minimum 2 years' observation in every patient has demonstrated late rupture and emboli to be important problems. The fascial valve does not retain normal stress-strain characteristics which would make it superior to other forms of biological valve. Sudden rupture of cusp tissue renders fascia lata valve replacement more hazardous than fresh homograft replacement where valve rupture is uncommon. One can only be guarded about the long-term prognosis, and centres looking for a new form of aortic valve replacement should not be guided by the initial favourable reports.

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References

Balcon, R., Emanuel, R., Lincoln, C., McDonald, L., Ross, D., and Somerville, J. (1970). Fascia lata replacement for isolated aortic valve disease. A follow-up of 23 cases. Abstracts, 6th World Congress of Cardiology, London. *Cardiovascular Research*, p. 70.

British Medical Journal (1970). Epidemiology notes. **2**, 185.

Dalichau, H., Gonzalez-Lavin, L., and Ross, D. N. (1972). Autologous fascia lata transplantation for heart valve replacement. *Thorax*, **27**, 18.

Edwards, W. S. (1969). Aortic valve replacement with autogenous tissue. *Annals of Thoracic Surgery*, **8**, 126.

Edwards, W. S., Karp, R. B., Robillard, D., and Kerr, A. R. (1969). Mitral and aortic valve replacement with fascia lata on a frame. *Journal of Thoracic and Cardiovascular Surgery*, **58**, 854.

Gonzalez-Lavin, L., and Ross, D. N. (1971). Autologous fascia lata valves in combined aortic and mitral valve surgery. *Annals of Thoracic Surgery*, **12**, 236.

Ionescu, M. I., Deac, R. C., Whitaker, W., and Wooler, G. H. (1972). Fasica lata heart valves. In *Biological Tissue in Heart Valve Replacement*, p. 617. Ed. by M. I. Ionescu, D. N. Ross, and G. H. Wooler. Butterworths, London.

Ionescu, M. I., and Ross, D. N. (1969). Heart-valve replacement with autologous fascia lata. *Lancet*, **2**, 335.

Ionescu, M. I., Ross, D. N., Deac, R., Grimshaw, V. A., Taylor, S. H., Whitaker, W., and Wooler, G. H. (1970a). Autologous fascia lata for heart valve replacement. *Thorax*, **25**, 46.

Ionescu, M. I., Ross, D. N., Deac, R. C., and Wooler, G. H. (1970b). Heart valve replacement with autologous fascia lata. *Journal of Thoracic and Cardiovascular Surgery*, **60**, 331.

Petch, M., Somerville, J., Ross, D., Ross, K., Emanuel, R., and McDonald, L. (1974). Replacement of the mitral valve with autologous fascia lata. *British Heart Journal*, **36**, 177.

Ross, D. N. (1967). Homograft replacement of the aortic valve. Technique. *British Journal of Surgery*, **54**, 165.

Ross, D. N., and Somerville, J. (1971). Fascia-lata reconstruction of the right ventricular outflow tract. *Lancet*, **1**, 941.

Senning, A. (1967). Fascia lata replacement of aortic valves. *Journal of Thoracic and Cardiovascular Surgery*, **54**, 465.

Senning, A. (1968). Results of fascia lata reconstruction of the aortic valve. *Journal of Cardiovascular Surgery*, Suppl., p. 28.

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